

**Atg13 (E1Y9V) Rabbit mAb**

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**For Research Use Only. Not for Use in Diagnostic Procedures.**

Applications:	Reactivity:	Sensitivity:	MW (kDa):	Source/Isotype:	UniProt ID:	Entrez-Gene Id:
W, IP, IF-IC	H	Endogenous	72	Rabbit IgG	#O75143	9776

**Product Usage Information****Application**

Western Blotting  
Immunoprecipitation  
Immunofluorescence (Immunocytochemistry)

**Dilution**

1:1000  
1:100  
1:100 - 1:200

**Storage**

Supplied in 10 mM sodium HEPES (pH 7.5), 150 mM NaCl, 100 µg/ml BSA, 50% glycerol and less than 0.02% sodium azide. Store at -20°C. Do not aliquot the antibody.

For a carrier free (BSA and azide free) version of this product see product #66925.

**Specificity/Sensitivity**

Atg13 (E1Y9V) Rabbit mAb recognizes endogenous levels of total Atg13 protein.

**Source / Purification**

Monoclonal antibody is produced by immunizing animals with a synthetic peptide corresponding to residues surrounding Asn230 of human Atg13 protein.

**Background**

Autophagy is a catabolic process for the autophagosomic-lysosomal degradation of bulk cytoplasmic contents (1,2). Autophagy is generally activated by conditions of nutrient deprivation but has also been associated with a number of physiological processes including development, differentiation, neurodegeneration, infection, and cancer (3). The molecular machinery of autophagy was largely discovered in yeast and referred to as autophagy-related (Atg) genes.

Atg13/Apg13 was originally identified in yeast as a constitutively expressed protein that was genetically linked to Atg1/Apg1, a protein kinase required for autophagy (4). Overexpression of Atg1 suppresses the defects in autophagy observed in Atg13 mutants (4). Autophagy requires a direct association between Atg1 and Atg13, and is inhibited by TOR-dependent phosphorylation of Atg13 under high-nutrient conditions (5). Similarly, mammalian Atg13 forms a complex with the Atg1 homologues ULK1/2, along with FIP200, which localizes to autophagic isolation membranes and regulates autophagosome biogenesis (6-8). mTOR phosphorylates both Atg13 and ULK1, suppressing ULK1 kinase activity and autophagy (7-9). ULK1 can directly phosphorylate Atg13 at a yet unidentified site, presumably to promote autophagy (7,8). Additional studies suggest that Atg13 and FIP200 can function independently of ULK1 and ULK2 to induce autophagy through an unknown mechanism (10).

**Background References**

1. Reggiori, F. and Klionsky, D.J. (2002) *Eukaryot Cell* 1, 11-21.
2. Codogno, P. and Meijer, A.J. (2005) *Cell Death Differ* 12 Suppl 2, 1509-18.
3. Levine, B. and Yuan, J. (2005) *J Clin Invest* 115, 2679-88.
4. Funakoshi, T. et al. (1997) *Gene* 192, 207-13.
5. Kamada, Y. et al. (2000) *J Cell Biol* 150, 1507-13.
6. Ganley, I.G. et al. (2009) *J Biol Chem* 284, 12297-305.
7. Hosokawa, N. et al. (2009) *Mol Biol Cell* 20, 1981-91.
8. Jung, C.H. et al. (2009) *Mol Biol Cell* 20, 1992-2003.
9. Kim, J. et al. (2011) *Nat Cell Biol* 13, 132-41.
10. Alers, S. et al. (2011) *Autophagy* 7, 1423-33.

**Species Reactivity**

Species reactivity is determined by testing in at least one approved application (e.g., western blot).

**Western Blot Buffer**

**IMPORTANT:** For western blots, incubate membrane with diluted primary antibody in 5% w/v BSA, 1X TBS, 0.1% Tween® 20 at 4°C with gentle shaking, overnight.

**Applications Key**

**W:** Western Blotting **IP:** Immunoprecipitation **IF-IC:** Immunofluorescence (Immunocytochemistry)

**Cross-Reactivity Key**

**H:** Human

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